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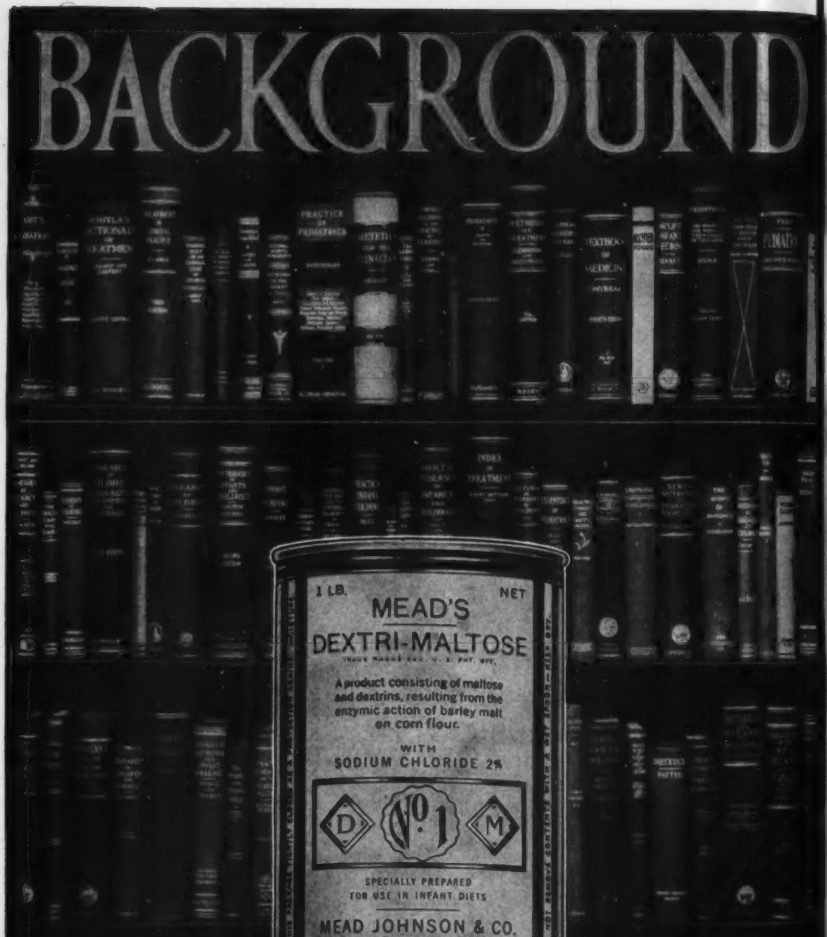

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SPECIAL REPORT

SURVEY OF POLIOMYELITIS CASES FOR 1948

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It is our purpose to present the clinical features and some investigational laboratory findings of poliomyelitis, based on the observation of patients admitted to the Children's Hospital in 1948 with a diagnosis of this disease.

There was a rather marked increase in incidence of reported cases of poliomyelitis throughout the nation this past year, reaching epidemic proportions in several sections of the country such as California, North Carolina and Texas, which together were afflicted with 35% of the total cases, as recently reported by the National Foundation. In the greater Washington area, there were 169 cases, representing a ratio of 18 per 100,000 population. This figure is comparable to the epidemic year of 1944 when there were 194 cases in the District, with a ratio of 21.5 per 100,000.

During the period from June 21 through November 29, there were 157 patients admitted to this hospital with a diagnosis of poliomyelitis. The majority of these cases occurred from the latter part of July through October with the peak of admissions in August.

Of the cases admitted, 61 or 38% were from the District of Columbia, 54 patients or 34% were residents of Virginia, 41 patients or 26% from Maryland and 1 patient contracted the disease shortly after his arrival in Washington from Louisiana.

There was a slight predominance of males over females, there being 92 cases of the former, 65 of the latter. This distribution is not unusual.

There was a marked distinction in the racial component in our group, there being 132 white patients in contrast to 25 colored. This predominance of the number of white to colored population does not readily lend itself to any conclusion since there are no available figures for the proportion of white to colored population in the large geographical area from whence our patients originated. It is interesting to note that the poliomyelitis survey for this area in 1944 showed a similar ratio of 6 white patients to one colored.

Examination of the recorded histories of these patients revealed the following interesting data. In 109 or 70% of the cases, there was either no definite history of contact obtained or in some cases no notation of such was made. In 38 instances, or 24%, it was noted that the patient had been exposed to some vague syndrome with such symptoms as fever, malaise,

headache or signs of upper respiratory infection. Such a syndrome may be adjudged to be of suspicious contact. Ten cases, or 6%, were admitted with a definite history of exposure to a known case of poliomyelitis. Of these, eight were instances of siblings with obvious intimate contact. Taking the latter two groups together, those with a suspicious and those with a positive contact, we have a total of 48 cases, with a percentage of 30.5. Most investigators report that a positive history of contact is usually noted in 10 to 15% of cases. Casey of Alabama reports a positive contact history in 80% of cases. This high figure, however, has not been duplicated by other investigators. It should be borne in mind that such a high contact history can only be obtained after painstaking interviews, home visits and other such follow-up questionnaire methods.

There was nothing unique in the symptomatology exhibited by our group of cases. It might be well, however, to mention the cardinal symptoms as we found them, that in a poliomyelitis season presuppose to the latter diagnosis. These were in order of their frequency (1) low grade fever, (2) headache, (3) stiffness of the neck, (4) gastrointestinal disturbances such as nausea or vomiting and (5) spasm or weakness of the lower extremities. Three patients presented a history of a major convulsion prior to entry, the latter being a rather infrequent finding.

Analysis of the figures according to the types of poliomyelitis, reveal 133 cases, or 85%, classified as the spinal form; 11 patients, or 7%, as pure bulbar and 13 cases, or 8%, with bulbar-spinal involvement, making a total of twenty-four cases with some form of bulbar manifestations. At a superficial glance, the incidence of bulbar involvement appears to have increased in frequency as compared to earlier periods such as the terrible epidemic year of 1916, when proportionately fewer bulbar cases were reported. However, this increase in number probably does not represent an actual increase in frequency. The most likely explanation is the general establishment of criteria for the diagnosis of bulbar poliomyelitis. Today the term "bulbar" includes all those cases with involvement of the cranial nerves. This appreciation of the diagnosis of bulbar poliomyelitis should be given wider publication to the layman, in that every case of bulbar is not as serious as the general acceptance of the term seems to imply. For the purpose of emphasis, let us review the cases of bulbar and bulbar spinal poliomyelitis for the past year. Of the 11 pure bulbar cases, there were but four with respiratory center involvement and three of these were fatalities. Of the remaining seven, five had isolated facial nerve palsy, one with palate weakness and nasal twang and one patient whose only involvement was difficulty in swallowing. Of the thirteen cases of bulbar-spinal poliomyelitis, there were just two instances of intercostal nerve paralysis, one of them requiring the use of a respirator for a short period and the other accommodating himself

to his difficulty. In retrospect, of the twenty-four patients with bulbar involvement, eighteen exhibited mild symptomatology with a usual course of rather prompt improvement.

Evaluation of the individual clinical pictures in the three fatal cases revealed in the first instance a seven year old white boy admitted with a history of a severe headache, vomiting and a low grade fever of two days duration. The patient presented a toxic appearance and moderate stiffness of the neck on admission. Spinal fluid examination revealed 165 cells per cu. mm. with 69% polys and 20 mgms. % of protein. The patient gradually became comatose with a rising temperature and development of peripheral circulatory collapse and he expired 32 hours after admission. The second case is that of a six year old white girl who complained of headache, low grade fever and difficulty in swallowing of several days duration. Marked neck and back spasm with right facial nerve paralysis were noted on admission. Lumbar puncture was not performed. The patient was started on Darvisul treatment on the second hospital day, receiving the drug intravenously for six days. She was administered parenteral feedings for the first six days, then changed to feedings by gavage. No clinical improvement was noted and on the eleventh day the child developed progressive air hunger and expired twenty-four hours later. The third case was that of an eight year old white girl with a history of headache, vomiting and drowsiness of two days duration. On admission the patient exhibited a nasal twang, facial nerve paralysis and marked nuchal rigidity. Spinal fluid examination revealed 176 cells per cu. mm., 74 neutrophiles and 40 mgms. % of protein with normal sugar value. Immediately after admission, the patient developed marked respiratory distress with cyanosis and severe substernal retraction. Bronchoscopy and tracheotomy were performed but the patient failed to improve. She developed progressive circulatory collapse accompanied by irregular respirations and auscultatory evidence of auricular fibrillation. She was placed in a respirator because of almost complete cessation of breathing, but failed to respond, expiring 36 hours after admission.

At this point we should like to emphasize that our attitude concerning the use of the respirator is quite conservative and the child placed in such an apparatus is done so only after critical evaluation.

Spinal fluid findings in this disease are well known and require little repetition except to mention that the degree of abnormality in the spinal fluid apparently bears no relationship to the degree of involvement. It may be well to mention a few unusual features noted in several of our cases. The cell count was normal in ten instances, using six cells as the upper limit of normalcy. The highest cell count was 1,100 per cu. mm. Normal protein values were found in fifty-one cases using 20 mgm. % as the upper limit of normal. Differentials were of no particular significance.

The physical evaluation of the patients on discharge from the hospital revealed that 71 cases, or 46%, had no apparent residual defect. Of this number how many will ultimately be considered free of involvement will be decided only after a lengthy period of observation. There were forty-four cases, or 28%, who exhibited a moderate degree of paralysis. In this category we have included weakness of an extremity without loss of function, nasal twang, or some mild difficulty in swallowing. There were thirty-nine cases, or 24%, with a severe degree of paralysis ranging from loss of complete function of one extremity to the more severe case involving paralysis of several muscle groups. There were three deaths which were discussesd elsewhere.

An appraisal of the incidence of the disease and incidence of residuals according to age presents some interesting data. There were twenty-five cases under the age of two years, representing 15% of the total, the youngest being six months. In this group, only in one instance was there recovery without any apparent residual, representing a rate of 96%.

There were fifty-seven cases occurring between the ages of two and six years representing 36% of the total cases. Twenty-eight of these patients were found to have residuals at the time of their discharge, giving an incidence of residuals for this age group of 50%.

The third group between the ages of six and fourteen years included the remainder of the patients. There were seventy-five cases in this group, 47% of the total number. Thirty-one of these patients were found to have residuals on discharge, this representing an incidence of residuals of 45%.

The interpretation of these figures suggests the possibility of at least two factors sharing the responsibility. First, examination of the histories in the infant group reveals that the majority of these patients showed flaccid paralysis as the first or at least a relatively early symptom of their illness. From this it would appear that the prodromal symptoms were less conspicuous in this age group. One might speculate that if these prodromal symptoms such as headache, nausea, nuchal and extremity spasm and backache went unnoticed in most of these cases with flaccid paralysis, then it might be that they were inconspicuous in a theoretical non-paralytic group which went undiagnosed. If this theory be correct, the explanation for the seemingly high percentage of residuals in the infant group would be apparent.

The second factor could be that the central nervous system in infancy is more likely to have pathological changes resulting from infectious disease involving the central nervous system than in the older age group. There seems to be a correlation between the clinical residuals according to age groups in poliomyelitis and in pyogenic meningitis insofar as the residual rate is highest in the infancy group. This is supported by studies carried on in this institution.

The period of hospitalization varied directly with the degree of involvement. From the overall picture we may appreciate some encouraging facts which might well be emphasized to the lay public. There were fifty-eight cases which were hospitalized for a period of less than two weeks; forty-five cases between two and four weeks; thirteen cases between four and six weeks; sixteen cases between six and eight weeks; four cases between eight and ten weeks and ten between eight and twenty-two weeks. At the present time, there are four patients remaining on the wards. As stated above, these are encouraging figures in that one hundred and three cases were discharged from the hospital in less than one month, this group including the seventy-one non-paralytic cases, the other thirty-two cases having only moderate involvement.

Electrocardiographic studies were performed on forty-four patients. Thirty-four of these patients had normal tracings and ten cases, or 22% showed abnormalities. Our procedure in this group was to do an initial electrocardiogram on admission and to repeat it in two weeks. Those patients having abnormal findings were examined at two week intervals until tracings were normal. In all but one instance, the abnormal electrocardiographic findings have reverted to normal.

The electrocardiographic changes consisted of temporary prolongation of the P-R interval in one patient, flattening of the T waves in Leads 1 and 2 in another patient, and in the remaining eight patients there was depression of the S-T segment and diphasic to inverted T waves in Leads 2 and 3. Unipolar and chest leads were not done in any of the patients.

The abnormal electrocardiograms suggest that the virus of poliomyelitis is distributed, in the affected patient, to the heart as well as to the central nervous system. The fact that the electrocardiographic changes were confined to Leads 2 and 3 in the majority of the patients suggests that unusual strain on the right side of the heart due to increased intrapulmonary pressure or some clockwise rotation of the heart on its vertical axis was responsible for these abnormalities rather than myocarditis. It has been shown that in many patients dying of this disease, histologic changes in the heart muscle indicative of myocarditis have been found. These changes were recognized for the first time by Saphir and Wile in 1942. Lucchesi in 1943 and Gefter et al. in 1947 described electrocardiographic changes in living patients similar to those we have found in our patients and diffused myocarditis on microscopic examination in several fatal cases.

Clinical examination has not led to suspicion regarding heart involvement in any of the patients in this series or in those reported by others. It appears then, as in many other diseases, that the heart involvement here is the result of widespread infection and if the individual survives that infection, there is no residual heart damage. However, we should be on the watch for cardiac involvement during the clinical observation of the patient with poliomyelitis

and be ready to combat as far as possible cardiac dysfunction in the rare patient who will show it.

During the latter part of August, a limited supply of Sodium Phenosulfazole, (Darvisul) was made available through the courtesy of the Lederle Laboratories and the National Institute of Health. Since the drug was to be used in several other centers in the midst of major epidemics where clinical evaluation could be more readily appraised, it was decided that our study should be directed to its possible toxicity.

The dosage used was 400 mg. per kilo body weight per twenty-four hours. Its mode of administration was by intravenous route in continuous infusion of 5% glucose in water for the first 36–48 hours and then continued orally in the same dosage. Of the thirty-three patients receiving the drug, sixteen recovered from the disease without any apparent residuals, fifteen had residual paralysis and two died. Both fatalities were bulbar cases. One received the therapy for thirty-six hours, the other for six days. There was severe vomiting in three instances necessitating discontinuance of this medication. All three patients were older children requiring large doses of the drug. Mild leukopenia was noticed in some cases, but this was noted in many others not receiving Darvisul.

Our experience with Darvisul is in agreement with that of Dr. Clifford Grulee, Jr. from Texas who, in a carefully controlled series, concluded that this drug showed essentially negative results in the treatment of poliomyelitis.

In summary, we have presented the clinical features and some investigational laboratory findings based on 157 patients with a diagnosis of poliomyelitis during the past year.

Several of the prominent features were as follows:

1. There was a marked distinction of the racial component in our group with a predominance of the number of white over colored patients in a ratio of 5 to 1.

2. Ten cases, or 6%, gave a positive history of contact with another case of poliomyelitis. Thirty-eight instances were noted of exposure to a vague syndrome which may be adjudged to be of a suspicious contact.

3. There were twenty-four cases with some form of bulbar involvement, the majority of these exhibiting mild symptomatology with a rather prompt improvement noted. There were three fatalities in this group.

4. The incidence of residuals in the infant group consisting of patients from 6 months to two years was 96% or 24 of 25 cases.

5. One hundred and three cases, or 65%, were discharged from the hospital in less than one month. This figure may well be used to alleviate somewhat the fear and anxiety of parents during a poliomyelitis season.

6. Electrocardiographic studies were performed on forty-four patients.

Ten had abnormal tracings; in nine instances of this group, the tracings have reverted to normal.

7. Thirty-three patients received Darvisul treatment. Minimal toxicity to the drug was noticed. The treatment appeared to be of no value in poliomyelitis.

BIBLIOGRAPHY

1. Anderson, G. W.: Epidemiology of Poliomyelitis. Lancet, **67**: No. 1, p. 10, January 1947.
2. Saphir, O., and Wile, S. A.: Myocarditis in Poliomyelitis. American Journal Medical Science, **203**: 781–788, June 1942.
3. Luccesi, H.: Electrocardiographic Changes in Poliomyelitis. American Heart Journal, **33**: No. 1, p. 228, January 1947.
4. Gefter, W. I. et al: Heart in Atcue Anterior Poliomyelitis. American Heart Journal, **33**: 538–540, April 1947.
5. Casey, A. E.: Place of Contact and Radial Spread of Poliomyelitis. Am. J. Diseases Child., **69**: 152–156, March 1945.
6. Dauer, C.: Personal Communication. D. C. Health Dept.
7. Grulee, C., Jr.: Personal Communication. University of Texas, Galveston, Texas.

OTITIC HYDROCEPHALUS MENINGEAL HYDROPS

Case Report No. 156

Hassan Ahari, M.D. M. L. H. 44–2903

M. L. H., a six year old white female, was admitted to the Children's Hospital in February 1949 complaining of fever and infected ears. She was well until five days prior to admission when she started to have a fever, complained of ear ache, abdominal pain, and appeared to be drowsy. Two days prior to admission she vomited several times and complained of headache. On the day prior to admission she developed convergent strabismus. No medication was given during the period prior to hospitalization.

Past history revealed that the child was the product of a normal gestation, but due to a prolonged labor of 24 hours duration, a Caesarean section was performed. The infant weighed 3,390 gms., and the dietary intake during infancy was adequate. Routine immunization procedures for diphtheria, tetanus, pertussis, and smallpox were performed. Her growth and development were normal. At one year of age she contracted measles and at eighteen months of age a left inguinal herniorrhaphy was performed. During the previous two winters she developed several upper respiratory infections, some of which were associated with infected ears, and were treated with sulfonamides or penicillin.

The family history was not contributory.

Physical examination on admission disclosed a temperature of 101.4°F., respirations 25 per minute, pulse 98 per minute, and blood pressure 130/100. Both ear drums were injected and the pharynx and tonsils were inflamed. Mild cervical lymphadenopathy was present.

The pertinent neurological findings included nuchal rigidity, rapidly advancing papillodema (later with hemorrhages), left sixth nerve paralysis which 24 hours later involved the right abducens, limited upward gaze, and pyramidal signs with difference in patellar reflexes, possible Babinski on the left and right facial weakness. There were no cerebellar signs or visual field defects.

Spinal tap revealed crystal clear fluid with only one white blood cell and a total protein of 5 mgms. The pressure was 280 mm. water. All cultures were reported as negative. On the following day the spinal fluid pressure was 400 mm. water and bilateral occipital trephination was performed; 35 cc. cerebrospinal fluid was removed gradually from the right lateral ventricle. The fluid was under increased pressure but was essentially negative for cells and protein. This was fractionally replaced with 25 cc. of oxygen. A Frazier canula was left in the right lateral ventricle. X-rays of

ventricular air showed symmetrical ventricles which were normal in size. The iter was not visualized but its patency was assured by a small amount of air in the 4th ventricle and over the cortex. On the following day oxygen was introduced from below as well as through the Frazier canula without successfully outlining the aqueduct. The Lateral Ventricles actually appeared smaller than normal. This has been noted by others[8]. Antibiotics were resumed. Spinal taps were started while the Frazier canula was in place and continued daily when the canula was removed 48 hours later. The patient immediately improved and the stiff neck disappeared, but each day the fluid was found under high pressure ranging from 300 to 500 mm. water. She ate well and took fluids normally without vomiting or nausea.

An x-ray taken two weeks after admission showed a mastoiditis. A right mastoidectomy was performed on the 9th of March, 1949 and an abscess was encountered. The dura was covered with granulations and the lateral sinus was bulging but easily compressible indicating it was not thrombosed. (Quickenstadt test was normal several days later on brief compression of each jugular.) Her post-operative course was uneventful and she continued to improve although the cerebrospinal fluid pressure was still elevated (ranging from 220–280 mm. water). On the 5th post-operative day only a minimal 6th nerve paresis remained on the right. There was no limitation to upward gaze and the pyramidal signs had disappeared. A left mastoidectomy was done one week later and chronic infected cells encountered. Culture of this material was negative. Convalescence again was uneventful and improvement continued slowly. Spinal taps were done every 1–2 days and the papillodema gradually decreased from 4–5 diopters to 2 in the right and ½ in the left at the time of discharge on April 4th, 1949. The spinal fluid pressure at this time was 140. On leaving the hospital she was alert, happy, and the remaining neurological examination was entirely negative.

When seen in the office two weeks after discharge she was well and happy and completely asymptomatic. Neurological examination was negative except for a one diopter papillodema in the left eye. Vision was 20/20 in each eye. Ayala index was 8.

DISCUSSION

William Thornton Spence, M.D.: This is a report of an early case of Otitic Hydrocephalus that developed under treatment for otitis media. It conforms in all details to the criteria established by Symonds[1] who gave it the above name.

The purpose of the report is to emphasize that this condition, once started, progresses in spite of current antibiotics which culturally sterilize the encapsulated pus (mastoids in this case). A procedure of diagnosis and treatment is given.

By definition the condition of Otitic Hydrocephalus, or, what seems like a better term, Meningeal Hydrops, is increased intracranial pressure due to excessive production of *normal* cerebrospinal fluid or failure of it to be absorbed. It always seems to follow infection, especially otitis media or mastoiditis.

Review of the literature indicates that specific mention of Otitis Hydrocephalus has occurred about 15 times since 1931. The number of cases varied from 1 to 15. Since 1941 the condition has been mentioned in the literature on only four occasions and of these only two were case reports. The assumption is that fewer cases are found since the advent of antibiotics but perhaps the differences lie in nomenclature. Kinnier Wilson[2] does not list Otitic Hydrocephalus but appears to include it in meningitis serosa without indicating it as a distinct clinical entity. Yet he specifies that all types of meningitis serosa include an elevation of spinal fluid protein or cells or both, which increase is consistently lacking in the case reported and by others describing this syndrome. Undoubtedly many cases are treated as meningitis or under some other diagnosis. The first concise article bearing the title Otitic Hydrocephalus was that of Symonds[1] in 1927 and later articles[3] one of which[4] described the additional finding of lateral sinus thrombosis. Leidler[5], on the strength of a single case, feels that the condition may result from lateral sinus thrombosis. Partial venous sinus thrombosis is held to be the cause by Davidoff[6] who also states that he is still seeing the condition and treating it by decompression. Symonds points out, however, that if this were true it would be a frequent accompaniment of venous sinus thrombosis instead of an accepted rarity[4]. In our case, as in many others in the literature, there was no thrombosis of the lateral sinuses. The tenseness of the right lateral sinus at operation seemed due to distended venous sinus from venous engorgement but may have been in part due to pre-cerebellar accumulation of cerebrospinal fluid as described by Bing, Baramy, and Jenkins[7]. In fact, such an accumulation in the superior cistern exerting pressure on the quadrageminal plate might explain the limitation of upward gaze. Or if the great vein of Galen were distended to the same extent as the lateral sinus it is conceivable that it could press on the superior calliculus and account for the limitation of upward gaze. It may be surmised compression of this vessel alone from a distended superior cistern might play the principal role in producing the entire syndrome.

The cases reported describe the condition as existing from six weeks to two years. In a series of fifteen cases reported over a five year period at the Neurological Institute, New York City by Davidoff and Dyke[8] the average duration of time before diagnosis and treatment was instituted ranged from 12 days to 6 months. The case herein reported developed under observation; only blurred discs were noted on first examination, yet full-blown

papillodema was present two days later and the signs of 6th nerve weakness, positive Babinski, etc., also followed two days after the first vomiting and drowsiness. This case serves to indicate what the time factor may be in the development of this condition and calls attention to the fact that therapy must be instituted early if eyesight is to be preserved. Since the most serious sequella is optic atrophy it would be interesting to know how many cases of adult atrophy could be traced to Otitic Hydrocephalus in their youth. Walsh[9] discusses the condition but does not include analysis of such a group. All ophthalmoscopic findings in the case reported are entirely negative except slight haziness of the left disc margin 5 weeks after discharge from the hospital. Her vision is normal.

Treatment started with a *diagnostic* ventriculogram and from that time on the cerebrospinal fluid pressure was never allowed to remain constantly elevated. The air study indicated an open system with a failure of the aqueduct to outline even though oxygen was simultaneously injected into the ventricle and subarachnoid space. A Frazier canula was placed in the occipital horn of the lateral ventricle for 24 hours prior to withdrawing fluid by the lumbar route to first decompress from above. This was a safety precaution which may not have been necessary but one which seems justified in view of papillodema and stiff neck even though cerebellar signs were lacking. These are often lacking in midline tumors and at the time of the ventriculogram this child resembled a brain tumor in many respects. Brain abscess was not considered likely with one cell and 5 mgms. protein in the spinal fluid on three examinations in 24 hours. Encephalitis and meningitis were also ruled out in view of the negative spinal fluid findings. Atresia of the aqueduct and tumor of the brain stem were eliminated by the air studies. Other conditions such as pineal or vermia tumor, parasitic or congenital cysts, etc. were ruled out by ventriculogram also. The facts indicate a toxic factor (otitis media in this case) set off the process of increasing the cerebrospinal fluid or inhibiting its absorption. The Ayala index ranged from 20 in the early period to 8 on discharge from the hospital and was 6 two weeks later. In a general way it seemed to follow improvement of cerebrospinal fluid tension. The composition of cerebrospinal fluid was normal at all times except for a cellular reaction following ventriculogram. The protein, sugar, and cells were lower than normal. This decrease included the chlorides (640–660) until recovery when they came back to normal (725 mgms.). This is of interest since study of the cerebrospinal fluid chlorides was suggested by Greenfield[10] who considered them of primary importance in gauging damage to the cerebrospinal fluid mechanism. By his theory the chlorides in this case would indicate a wide damage to the diffusion area to allow them to pass back into the blood. It would not hold, however, that this disturb-

ance was on a meningeal infection basis as he postulates. No other studies on chlorides in this condition could be found in the literature.

On pneumoencephalogram done *after* the condition had subsided (March 28th, 1949) the ventricles were of normal size, shape and position. This indicates to me that the ventricles are *smaller* than normal in the acute stage due to massive edema of brain tissue and that they revert to normal size at the end of the disease.

SUMMARY

1. A classical case of so-called Otitic Hydrocephalus is reported which did not respond to antibiotics.

2. Symptoms of increased intracranial pressure were seen, studied, and treated three days after they appeared.

3. Ventriculogram and ventricular drainage served to exclude other conditions and aided safely instigating spinal punctures which, with removal of focal infection, resulted in apparent cure.

4. Restriction of upward gaze in this condition is reported for the first time.

5. The Ayala index was estimated, composition of cerebrospinal fluid was followed including chlorides.

6. Pneumoencephalogram was done after the condition subsided and was normal in every respect.

7. The mechanism of Otitic Hydrocephalus is a complex physiopathological process which is not entirely understood. Meningeal Hydrops is suggested as a better term[11].

Note: When re-examined on May 6th, 1949, the child was still asymptomatic. Neurological examination was normal and optic fundi gave no indication of papillodema. Spinal fluid pressure was 145 mm. water; there were 4 cells and the Ayala index had come down to 2.

REFERENCES

1. Symonds, C. P.: Proc. Royal Soc. Med. Land., **20:** 1927, 1139.
2. Wilson, Kinnier: Diseases of the Nervous System, pp. 38–46.
3. Symonds, C. P.: Otitic Hydrocephalus, Brain, 1931, **54:** 55–71.
4. Symonds, C. P.: Otitic Hydrocephalus, Brit. Med. Journal, **1:** 53–54, 1932.
5. Leidler: Journ. Laryng. and Otol., **43:** 682, 1928.
6. Davidoff, Leo M.: Personal communication.
7. Bing, Baramy and Jenkins: J. Laryng. and Otol., **37:** 1922.
8. Davidoff and Dyke: Journ. Nerv. and Mental Diseases, **83:** 700, 1936.
9. Walsh, Frank: Otitic Hydrocephalus Serous Meningitis, 578–580, 1947.
10. Greenfield: The Chemistry of the Cerebrospinal Fluid in Otitic Meningitis. J. of Laryng. and Otol, **41:** 785–789, 1926.
11. Reilly, Henry Alsop: Personal Communication.

EVALUATION OF LABORATORY DATA OBTAINED FROM PROVEN CASES OF JAUNDICE

Robert O. Warthen, M.D.

This report constitutes an analysis of laboratory data from eleven cases of retentive jaundice and seventeen cases of complete obstructive jaundice encountered at the Children's Hospital over a thirteen year period. Unfortunately the series is small inasmuch as it is limited to those cases (a) with laboratory data pertaining to jaundice and (b) whose diagnoses were established by microscopic examination of liver tissue obtained either at surgery or necropsy. Hence the results obtained are merely suggestive. Laboratory data pertaining to jaundice were extracted from the charts reviewed and are tabulated in Tables I and II entitled retentive jaundice and complete obstructive jaundice respectively.

Table I, which comprises the retentive group, includes the following cases; one syphilitic hepatitis, three portal cirrhoses, one juvenile biliary cirrhosis, three cases of erythroblastosis foetalis, one jaundice of prematurity, and two cases of infectious hepatitis, retentive phase. In comparing the data derived from these cases with a classical textbook picture of retentive jaundice (Table I), we noted the following: Urobilinogen determinations or repeated urinalyses offered the most consistent reliable results, whereas bilirubin determinations on the urines and stools were reliable to a lesser degree; the qualitative van den Bergh seemed to be of very little value, for 80% of the tests performed yielded immediate and direct reactions, rather than the expected indirect or biphasic reaction. (It is to be noted that all of the hemolytic jaundice cases studied were fatal and were accompanied by varying degrees of liver damage which may have accounted for the immediate and direct reports, although even in such instances one would rather expect a biphasic reaction.) Icterus index and blood bilirubin levels correspond fairly closely with the classical picture; liver function tests were of aid mainly in establishing the presence or absence of hepatic cellular damage. Although stool urobilinogen determinations were not obtained in this series of retentive jaundice cases, it is the author's contention that they are equally as important as urine urobilinogen determinations.

Table II, which constitutes the complete obstructive group, includes the following cases; fifteen atresias of the bile duct, ten with secondary biliary cirrhosis and one with a choledochal cyst and two cases of infectious hepatitis—obstructive phase. A comparison of the data obtained from these cases with data expected in a classical textbook case (Table II) led to the following conclusions: Qualitative stool bilirubin determinations were 91% accurate, whereas urine urobilinogen and bilirubin findings were less depend-

TABLE 1

Retentive (Hepatic-Hemolytic) Jaundice

CASE NO.	DIAGNOSIS	STOOL				URINE				VAN DEN BERGH	BLOOD BILIRUBIN	ICTERUS INDEX	MISCELLANEOUS
		Urobilinogen		Bilirubin		Urobilinogen		Bilirubin					
		No. tests	Results	No. tests	Results	No. tests	Results	No. tests	Results				
											mgm. %	*units*	
45-4820	Syphilitic hepatitis									Immediate direct 1+	15	95	Kahn—+
41-4644	Portal cirrhosis			16 1 5	+ trace 0	22 2	+ 0	26 2	+ 0	Im. dir. 8+ Biph. 3+	2.2-8.0	25-200	Cephalin flocculation 4+ (48 hrs.). Prothrombin time 22-80% normal. Sedimentation rate 22-48. Cholesterol 100 mgm. %. Platelets 50,000
47-8448	Portal cirrhosis							2	+	Im. dir. 1+	1.6	33.8	
42-1991	Portal cirrhosis					2	0	3 1 tr	0 trace	Im. dir. 1+	2.6	39-53	Prothrombin time 61% normal.
42-4674	Juvenile biliary cirrhosis							1	+	Im. dir. 1+	14.2	200	Cephalin flocculation 3+ (48 hrs.)
45-7507	Erythroblastosis fetalis			2	+					Im. dir. 1+	12		
35-1600	Erythroblastosis fetalis							1	+	Im. dir. 1+		17	
44-6695	Erythroblastosis fetalis			1 2	+ 0			1	+	Im. dir. 1+	3.75	48-120	
47-9360	Prematurity									Biph. 1+	3.9		
46-3606	Hepatitis, retentive phase			2 1	trace +			1	0			84	
44-1125	Hepatitis, retentive phase					2	+	1	+	Im. dir. 1+	3.1	74	Sedimentation rate—20

Sum total of our findings in retentive jaundice		20 3 7	+ trace 0	24 4	+ 0	32 1 6	+ trace 0	Im. dir. 10+ Biph. 4+	1.6–15.0	17–200
Per cent of sum total of our findings in retentive jaundice		67% 10% 23%	+ trace 0	86% 14%	+ 0	82% 3% 15%	+ trace 0	Im. dir. 80% Biph. 20%		
Classical findings in retentive jaundice	+ to incr.		+ to incr.		+ to incr.		variable	Biph. or Indirect	1.5–15.0	15–300

+ = positive, 0 = absent, Im. dir. = immediate and direct, Biph. = biphasic.

TABLE II

Complete Obstructive (Regurgitant) Jaundice.

CASE NO.	DIAGNOSIS	STOOL				URINE				VAN DEN BERGH	BLOOD BILIRUBIN	ICTERUS INDEX	MISCELLANEOUS
		Urobilinogen		Bilirubin		Urobilinogen		Bilirubin					
		No. tests	Results	No. tests	Results	No. tests	Results	No. tests	Results				
											mgm. %	*units*	
45-4137	Atresia of bile duct Biliary cirrhosis			9 4 2	0 trace +			4	+				
45-2501	Atresia of bile duct			3 1	0 +			3 1	+ 0	Im. dir. 2+	5.1	60-102	
46-9789	Atresia of bile duct Biliary cirrhosis			2	0					Biphasic 1+	2.9	25	Ceph. floc. 4+ (48 hrs.)
44-5078	Atresia of bile duct Biliary cirrhosis			14	0	2	0	3 1	0 trace	Im. dir. 7+	1.4-6	20-120	Ceph. floc. 3+ (48 hrs.) Prothrombin 41-50% normal
47-5558	Atresia of bile duct Biliary cirrhosis			8	0	1 3	+ 0	1 2	+ 0	Im. dir. 1+	5.5	80	Prothrombin 60% normal. Ceph. floc. 2+ (48 hrs.) Cholesterol 170 mgm. % Alkaline phosphatase 44.4 King-Armstrong units
44-8258	Atresia of bile duct			5	0	1	0	2	0	Im. dir. 1+	0.8		
47-8884	Atresia of bile duct	5	0	5 4 5	+ trace 0	2 1	0 trace	1 4	+ trace	Im. dir. 3+	3.8-5		Ceph. floc. 4+ (48 hrs.)
41-10890	Atresia of bile duct			1	0					Im. dir. 1+ Biphasic 2+	7.2-8.7		Prothrombin 64% normal
40-612	Atresia of bile duct	1	0					1	0	Biphasic 1+		40	Prothrombin 61% normal

40–612	Atresia of bile duct Biliary cirrhosis	1	0					1	0	Biphasic	1+		40	Prothrombin 61% normal
47–7577	Atresia of bile duct Biliary cirrhosis	1	0	3	0	2 3	0 trace	5	+	Im. dir. Biphasic	1+ 1+	3.3–4.7	105–120	Ceph. floc. 3+ (24 hours)
44–8893	Atresia of bile duct Biliary cirrhosis									Im. dir.	1+	8.25	73	Prothrombin 29% normal
44–4550	Atresia of bile duct Biliary cirrhosis			2	0			2 1	0 +	Im. dir.	1+	5.4–12.6	55–108	Prothrombin 44–64% normal
45–588	Atresia of bile duct Biliary cirrhosis			3 1	0 trace			7	+	Im. dir.	2+	5–9.2	85–135	
43–3971	Atresia of bile duct			4	0	2	0			Im. dir.	1+	10.8	85–92	Prothrombin time 59% normal
46–8774	Atresia of bile duct Choledochal cyst Biliary cirrhosis			3	0	1	0	1	+	Im. dir.	1+	4.3	80	Ceph. floc. 2+ (48 hr.)
46–3606	Hepatitis—obstructive phase			4	0					Im. dir.	1+	2.2	56	Ceph. floc. 4+ (24 hrs.) Alk. phosphatase 24.1 K-A u.
44–1125	Hepatitis—obstructive phase			1	0			2	+	Biphasic	1+	5.7	25	
Sum total of findings (complete obstructive jaundice)		7	0	67 9 8	0 trace +	13 4 1	0 trace +	25 5 11	+ trace 0	Im. dir. Biphasic	23+ 6+	1.4–12.6	20–135	
Percentage of sum total—our findings in complete obstructive jaundice		100%	0	80% 11% 9%	0 trace +	72% 22% 6%	0 trace +	61% 12% 27%	+ trace 0	Im. dir. Biphasic	79% 21%			
Classical findings in complete obstructive jaundice			0		0 to trace		0		+ to incr.	Im. Dir.		up to 70.0	50–325	

able; the qualitative van den Bergh was 79% accurate; blood bilirubin and icterus index findings correspond favorably with the classical instance and liver function tests were of value in determining the presence or absence of liver cell damage. It is important to note that the stool urobilinogen data obtained was based on qualitative and not quantitative examinations. Had the more sensitive quantitative tests been performed on complete obstructive jaundice stools, one might have been misled by the findings of low stool urobilinogen levels. This may be explained by the fact that when high blood bilirubin levels are present, small amounts of bilirubin are passed from the blood stream into the intestinal tract with resultant conversion into traces of urobilinogen detectable by quantitative but not by qualitative tests; hence, the preference for qualitative determinations to eliminate confusion.

LIPODYSTROPHY

Case Report No. 157

Bjorn Gudbrandsson, M.D. E. B. F. 48–4518

E. B. F., a nine year old white female, was admitted to the Children's Hospital on April 17, 1948 with the chief complaint of wasting and pallor of the face. Three years prior to admission the parents noted that her face looked pale and thin. This had become gradually and progressively worse and she did not seem to thrive in spite of ostensibly good health and appetite. Six weeks prior to admission she developed mumps and a complicating otitis media had supervened with the production of an aural discharge during the week prior to admission.

The birth and developmental history were normal. She had had pneumonia two years before and had had frequent attacks of tonsillitis. Otherwise the patient had been very active and was doing well in school. Family history was non-contributory.

On physical examination the outstanding feature was the thin, pale, almost cadaverous looking face. There seemed to be complete absence of the subcutaneous fat in her face and neck and even the panniculus adiposus appeared to be gone. In contrast to this extreme wasting of the face, the trunk and extremities were moderately well developed. The subcutaneous fat in the extremities seemed to be present but not excessively so. There were no signs of muscular atrophy or other neuromuscular disorders. The tonsils were quite infected and both ears showed a mucopurulent discharge. The remainder of the physical examination was otherwise negative.

All laboratory examinations including a basal metabolic rate and blood cholesterol were within normal limits. X-rays of the chest, skull and long bones were similarly negative.

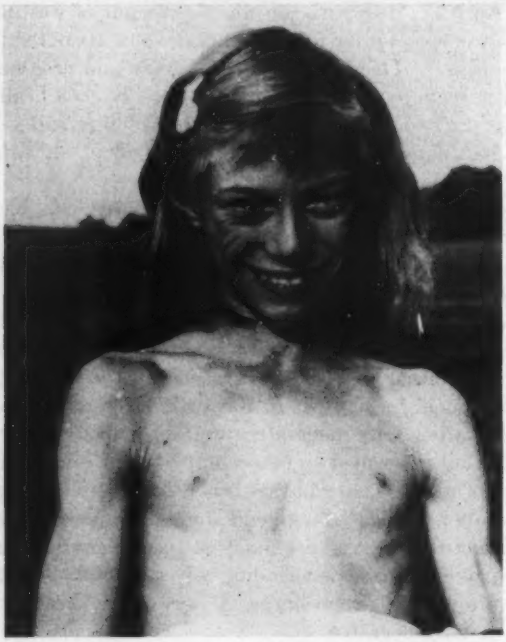
The patient was treated with penicillin and sulfadiazine for the draining ears and tonsillitis and she was discharged on the 23rd hospital day having recovered completely from the attack of otitis media. However, there was no demonstrable change in her features. At the present time, one year later, she is doing very well. Her face is unchanged but the process does not seem to have spread.

DISCUSSION

Lipodystrophy is a rare disease, approximately 95 cases having been reported in the literature to date. The disease is characterized by a complete loss of subcutaneous fat in the face and neck and occasionally the upper chest and upper extremities. In addition the panniculus adiposus in the

cheeks disappears which is of diagnostic value since this subcutaneous fat is the last one to disappear in an ordinary case of malnutrition.

In more than 75% of reported cases the disease started in childhood, mostly in or around the tenth year. The youngest case was two years of age. There is very little known about the cause of this disorder; some cases have started after repeated respiratory infections. The major theories are that it



Photograph by Frederick Fox

FIG. 1. E. B. F. This picture shows the characteristic features of the face and neck in lipodystrophy. (The picture was taken one year after the patient was discharged from the hospital.)

is a disturbance in the subcutaneous areolar tissue of the areas involved or that it is a disturbance of the trophic nerve mechanism which has to do with fat disturbance and fat metabolism. Biopsies of the affected areas have shown complete absence of fat beneath the corium. The appearance is quite different from that in extreme general emaciation where some fat always remains.

There is no specific treatment for this disease but the condition does not affect the general health. These cases are always a diagnostic problem in

view of their rarity. Any physician who has seen one case, however, will never have any difficulty in recognizing the picture again.

SUMMARY

A nine year old girl with lipodystrophy is reported with short review of the literature. The etiology of lipodystrophy is unknown and there is no specific treatment.

REFERENCE

1. Brenneman's Practice of Pediatrics. 1:41:1–7, iv:19:42–43, 1948.

CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.
Assisted by: D. Joseph Judge, M.D.
Edwin Vaden, M.D.
By Invitation: Claude Frazier, M.D.

Dr. Judge E. E. 45–619

E. E., a 20 month old white male child, was admitted to the hospital during the month of January with the probable diagnosis of pneumonia made by another physician.

Ten days before admission the child had an attack of asthmatic bronchitis which improved on medication. Two days prior to admission the child developed fever, lethargy and a respiratory grunt. During the week before admission, the child was seen to have become flushed and slightly cyanotic on two occasions during paroxysmal coughing episodes.

The birth and developmental history were normal. The feeding history was devoid of any significant findings. The child was apparently not allergic to any specific articles of his diet. The past medical history was negative except for one attack of croup at one year of age and infrequent intermittent asthmatoid wheezes associated with upper respiratory infection.

Physical examination on admission revealed a well developed, well nourished child in apparent respiratory distress. The alae nasi flared with inspiration and the abdominal musculature was used in respiration. Temperature was 103.6°F., pulse 116 and respirations 32. The other positive physical findings were as follows; mild injection of the pharynx, dullness to percussion over the left lower lobe posteriorly with depression of breath sounds in this area, rhonchi and wheezing coarse breath sounds throughout the remainder of the lung fields.

Initial laboratory work revealed 7.5 grams of hemoglobin with 2,480,000 erythrocytes; the leukocyte count was 10,700 of which 76 per cent were neutrophiles. Urinalysis showed 25 mgm. albumin with the remainder of the examination negative. An x-ray of the chest showed an increase in fibrosis and infiltration throughout the right chest. The left lung fields were clear.

Treatment was instituted and included sulfadiazine, fluids, and various symptomatic measures. The day after admission the child's temperature was 99.8°F. and it remained between 98–101°F. throughout hospitalization. The child continued to have episodes of coughing during which time he would perspire and flush. The original physical findings in the chest disappeared but the expiratory wheezes continued to be heard.

Fourteen days after admission the child suddenly developed a marked

inspiratory stridor with deep infra- and supra-sternal retraction. Examination of the lungs at that time revealed only the transmitted stridor. In a few hours the difficulty increased and tracheal intubation and constant oxygen was needed. Breathing stopped abruptly a short time after this, death occurring on the 15th hospital day.

DISCUSSION

Claude A. Frazier, M.D.: This twenty months old child had a history of frequent upper respiratory infections associated with wheezing. Such a history brings to mind the possibility of either asthma or congenital cystic fibrosis of the pancreas. The child also had a history of croup which occurs often in allergic children. However, there was no other allergic history (family or personal) nor was an eosinophilia present. No mention is made of the seasonal occurrence of the wheezing nor if any relief was obtained if adrenalin was used. In this age group the one major etiological factor of asthma is infection with onset of the asthma chiefly occurring in the winter months. In considering pancreatic cystic fibrosis the x-ray shows "fibrosis" in the right lung. Against this diagnosis, however, is the character of the stools, development and body build, all of which were apparently normal.

Ten days prior to admission the child developed what was called "an attack of asthmatic bronchitis." We do not know how suddenly this came on. It is very important in these cases to know the exact time of onset, whether the trouble began immediately after eating or just after vomiting and if at this time there was a paroxysm of coughing, choking, cyanosis or dyspnea. These symptoms would make one think of a tracheal or bronchial foreign body. The wheezing in these cases is due usually to the passage of air around the foreign body. The x-ray did not reveal a foreign body but this does not rule it out for if the seed or nut is translucent it cannot be visualized although the signs below the obstruction may be indicative of the etiological factor. The changes in the lung may vary as to the type of obstruction. The four types of bronchial obstruction are (1) by-pass valve obstruction in which air passes in and out of the lung involved in diminished amount, (2) a check valve obstruction in which air passes in and not out with resulting emphysema, (3) a reversed check valve obstruction in which air passes out and not in resulting in atelectasis and (4) shut valve obstruction in which air does not pass in or out resulting in atelectasis. It is important to take lateral films as well as AP films in any patient who wheezes not only as an aid in foreign body detection but to help eliminate the other causes which will be given later. Vegetal organic foreign bodies (peanut kernels, beans, etc.) are by far the most frequent and occur in this age group due to the undeveloped chewing technique. These vegetal organic substances cause an acute and early laryngotracheobronchitis with cough,

toxemia and fever differing from foreign bodies of bones, animal shells and inorganic bodies which usually produce all the symptoms of a chronic pulmonary sepsis, abscess and bronchiectasis with development of symptoms gradually over a period of months and years.

The child had seemed to improve with medication but then became worse two days prior to admission and was expectorating a considerable amount of white phlegm which makes one think of bronchiectasis. Bronchiectasis is a rare condition in asthmatic children but it is important to remember, however, that a patient with infective asthma producing large quantities of sputum, whose roentgenograms are entirely normal, should be examined bronchoscopically and if necessary enough lipiodol injected to outline clearly the bronchial tree. It is important to remember that a fair percentage of bronchiectases occur before the age of five years. Foreign bodies are likely to produce bronchiectasis by an incomplete obstruction of the bronchi with superimposed infection. The child had become cyanotic on occasion suggesting rather marked interference with oxygenation of the blood via the lungs and most likely not on a cerebral basis.

Physical examination revealed an ill child in respiratory distress. The temperature was elevated and the pharynx was injected. The physical examination was otherwise negative except for the chest which revealed depressed breath sounds in the left lower lobe with dullness to percussion in this area. Early in a foreign body case, diminished expansion of one side with dullness may suggest pneumonia. Also wheezes (supposedly expiratory) were heard over the remaining lung fields. "All that wheezes is not asthma." In differentiating asthma from some of the other causes of wheezing I might mention some of the routine diagnostic laboratory methods that we employ in Dr. Cook's allergy clinic in all cases of suspected bronchial asthma which are important diagnostic aids.* Other than a CBC, urinalysis and sedimentation rate, Schick and tuberculin tests are frequently indicated. A thorough ENT examination including a nasopharyngoscopic and laryngoscopic examination is done with a nasopharyngeal culture being taken at the same time. X-rays are taken of the nose, the paranasal sinuses, lateral x-ray of the soft tissues of the pharynx and an anterior-posterior and lateral view of the chest. If necessary a bronchoscopic examination with or without the instillation of lipiodol is done. Skin tests are routine. Occasionally such additional procedures as a BMR, electrocardiogram, venous pressure, circulation time, bleeding and coagulation time, platelet count, and vitamin assays are done. They occasionally uncover an unsuspected cause of the wheezing.

It might be well just to mention some of the other causes of wheezing

* Dr. Robert A. Cooke, Chief Allergy Department, Roosevelt Hospital, New York, N. Y.

not mentioned so far. Renal dyspnea in this case was most likely not a cause although a retinoscopy should have been done. There was a slight albuminuria on one occasion which was most likely due to the hyperthermia and supposedly was not found on other occasions. A pleural effusion could be present even though no signs were found by x-ray. Tuberculosis should be recognizable by x-ray and a tuberculin test would have been helpful.

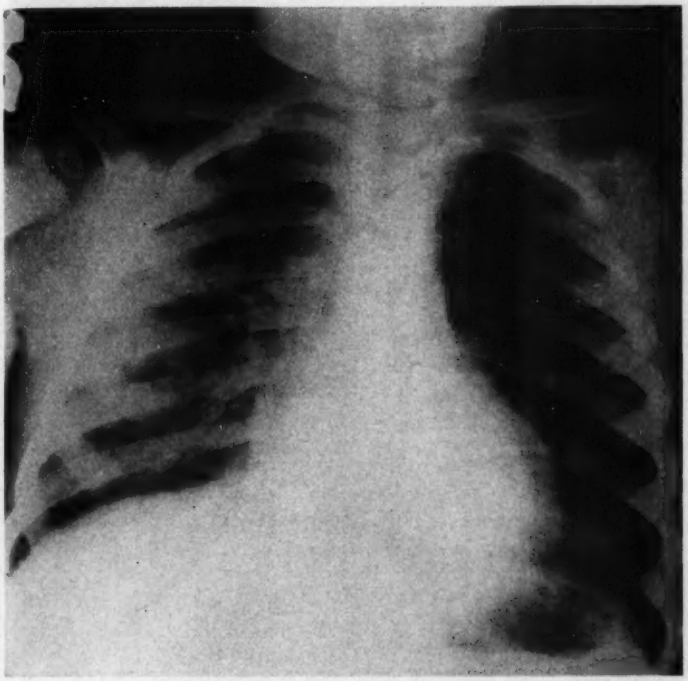


FIG 1: E. E. AP view of the chest showing slight eventration of the right diaphragm with scattered interstitial infiltration throughout the lower two-thirds of the right lung field.

Of course any form of bronchitis, tracheobronchitis or laryngotracheobronchitis, unspecific or specific, cannot be definitely ruled out and it is advisable to attempt to obtain cultures to determine the bacterial flora. Mediastinal growths and tumors, enlarged thymus, laryngeal papilloma or fibroma should be strongly considered as causes except that we are depending a great deal on the x-ray here to rule them out.

The x-ray revealed fibrosis of the right chest. Any case of localized fibro-

sis or a pneumonia which seems not to be clearing up should suggest lipoid pneumonia or a foreign body with or without bronchiectasis. Atelectatic areas of the lung may predispose to bronchiectasis with the indurated areas becoming fibrotic and tending to dilate the bronchi. There is no mention of other x-rays taken which is important in ruling out lipoid pneumonia. It gives a foreign body picture with the right side more often affected.

The hemogram revealed an anemia. There were no diagnostic studies undertaken to determine the cause. It is presumably in keeping with a chronic infection or malnutrition and is most likely not due to internal bleeding. There was only a slight elevation of the white blood cell count. This would seem to indicate either a low grade infectious process or lack of resistance on the part of the patient.

In following the child's hospital course we find that he was given sulfadiazine and symptomatic therapy. The original physical findings of the chest noted on admission seemed to change and thinking in terms of bronchial obstruction, bronchial asthma and acute bronchial infections, the most frequent causes of rapid shifting are bronchial obstructions.

Though the patient had episodes of coughing and ran a low grade fever, he was not in any apparent acute respiratory distress. However, on the fourteenth hospital day the patient suddenly developed inspiratory stridor. This suggests laryngeal involvement. Such involvement is frequently found in a foreign body of the larynx, papilloma, spasm of the glottis, laryngotracheobronchitis and laryngeal diphtheria. It would have been exceedingly helpful in this case to have had a Schick test, throat culture for diphtheria and a view of the larynx. Laryngeal angioneurotic edema as a result of sulfonamide sensitivity is possible as he had been on the drug long enough to develop the sensitivity but it is exceedingly rare. We find, however, that intubation did not relieve the symptoms and therefore the larynx alone must not have been involved unless the respiratory center had been damaged by anoxia or in a rare case where conceivably an intubation could force a polyp, foreign body, or laryngeal membrane into the bronchus.

We see then a precipitous sudden change for the worse in the child's condition with suprasternal retraction and only stridor transmitted. The sudden respiratory distress with absence of breath sounds is most likely due to massive atelectasis. The fact that intubation gave no relief would seem to indicate the trachea and perhaps the bronchi were involved as well. This involvement could be explained by a laryngotracheobronchitis, fluids or mucous in the trachea, tracheal secretions, a tracheal foreign body, a tracheal membrane, blood or pus in the trachea as may occur with rupture of an aneurysm, tumor or abscess into the trachea. Sudden respiratory distress with atelectasis may occur from a pneumothorax or pyopneumothorax from the rupture of a bronchus or abscess into the pleura and a possible

similar picture could be caused by acute right heart failure, but there were no other signs that would seem to indicate these as the cause and there would more likely be no suprasternal retraction.

We are left with several diagnoses which are very hard to discard and to decide on the final one which can give all the signs and symptoms as seen in this patient.

A rupture of an abscess or tumor into the trachea is to be considered but because of its rarity is to be ruled out.

Bronchial asthma seems unlikely even with the history of previous wheezing since there is really no other real evidence of allergy. It is exceedingly rare for a mucous plug to be coughed up and block the trachea to cause death. Asthma could very well be present and play a secondary role. In rare cases the patient may drown in his own secretions and occasionally develop bronchiectasis. Caffey states that the most frequent cause of bronchial obstruction is inflammatory disease of the bronchial tree.

The next three possible causes occupy almost equal importance in my mind. The x-ray picture might fit well into a lipoid pneumonia or aspiration class. Aspiration pneumonia could cover many things but if we think of lipoid pneumonia we find that on occasions the right side alone is involved as in this case. There is no characteristic sign or symptom. We need several films, however, to show the unchanging pictures. In its favor is the seemingly chronic course with low grade infection. The fatality rate is fairly high. Here it is possible for the patient to die by drowning in his own secretions. There should be a more accurate history of the infants oil intake (coughing, choking, cyanosis, etc.) to indicate aspiration.

Again, pancreatic cystic fibrosis could possibly give all of the symptoms and signs of this patient with x-ray findings of fibrosis in one lung. According to Dr. Anderson these children could die in a fashion similar to this patient either by right heart failure or drowning in their own secretions. Since we do not have an accurate history of stools or history of the other children we cannot definitely rule this out.

A tracheal or bronchial foreign body could very well account for all of the signs, symptoms and events in this case and cannot be excluded. Even the events requiring a tracheotomy can be explained by a violent tracheobronchitis which may occur with organic vegetal foreign bodies with edema in the subglottic region resulting in obstructions at this site. The one disconcerting part here is that we would expect the symptoms to be rather severe with high fever while in the hospital. Perhaps the sulfa therapy may have lessened the secondary infection. The location of the foreign body could be the bifurcation of the trachea as signs were present in both lung fields, perhaps blocking the right bronchus more at first giving the fibrosis or atelectatic areas and finally blocking the trachea or both

bronchi with massive atelectasis and death. If we could place the onset or intake of the foreign body ten days prior to admission it would most likely be due to a vegetal foreign body. If the croup at one year of age was taken as the time of onset, it would more likely be an inorganic substance, except it should be revealed by x-ray.

Finally, there seemed to have been a laryngotracheobronchitis. Whether it was a specific type as in H. influenza or diphtheria seems unlikely, and whether as a result of it there was a membrane or cast which involved the larynx and trachea terminally can be answered only by the pathologist. The laryngotracheobronchitis could have been produced by the foreign body and be part of the latter picture.

Final diagnoses:

1. Foreign body of the trachea—most likely organic vegetal.
2. Laryngotracheobronchitis—probably secondary to the foreign body.
3. Fibrotic and atelectatic areas of the lung.
4. Massive atelectasis of the lung.
5. Anemia—malnutritional.
6. Possible bronchiectasis, result of foreign body.

PATHOLOGICAL DISCUSSION

John E. Cassidy, M. D.: We are deeply indebted to Dr. Frazier for being with us and giving this most complete discussion and differential diagnosis.

As has been mentioned by Dr. Frazier and emphasized by many "all that wheezes is not asthma." This case is a very good example. For at autopsy there was found a peanut lodged at the bifurcation of the trachea completely obstructing it. A short distance down in the right main bronchus there was a depression in the mucosa, shaped like the peanut, and covered by a grayish inflammatory exudate. The lungs showed only small, irregular, scattered areas of atelectasis.

This patient had ingested and aspirated a peanut which lodged in the right main bronchus and produced the symptoms and signs mentioned. Death was caused by asphyxiation due to the foreign body being dislodged from the right main bronchus and lodging at the bifurcation of the trachea.

There is one matter of terminology which we would call to your attention. The words "increased fibrosis" have become a permanent fixture in the radiologists' vocabulary and are inadvertently transferred to pathological changes in the lungs which are not fibrotic. This is especially true in the disease cystic fibrosis of the pancreas. Changes occur in the lungs which are of an acute and chronic inflammatory nature with a minimum of fibrosis and that occurring late in the disease. It is true that the changes produced on the x-ray film appear to be similar to those produced by fibrotic changes but we must be careful to confine the term to radiologic appearances and not to pathological changes occurring in the lungs.